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OF

# PORTO RICO

## VARIOUS PAPERS ON THE INVESTIGATION OF SUGAR CANE DISEASES IN PORTO RICO

BY

JULIUS MATZ
*Chief Pathologist.*

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## GUMMING DISEASE OF SUGAR CANE.

By JULIUS MATZ.

### INTRODUCTION.

Gumming disease of sugar cane was noticed for the first time in Porto Rico in February 1920 in two car loads of sugar cane brought in from the neighborhood of the town of Trujillo Alto to be ground at Central Vannina, Río Piedras. Soon afterwards the distribution of the disease was ascertained by inspections of cane at the mills all over the Island, including the adjacent island of Vieques, and by inquiries from men engaged in sugar-cane cultivation. By the end of the harvest of 1920 the disease was located at the following places: Río Piedras; Guaynabo; south of the town of Trujillo Alto, between kilometers 13 and 18 along the Caguas Railway; at a point 9 kilometers from Río Piedras on the main highway going to Caguas; one gummed cane was found, at Central Juncos, in a last shipment of cane from the vicinity of Las Piedras; and another diseased specimen was seen in cane from near Morovis. This comprised the known infected locations at the end of the harvesting season of 1920. During that year the first observations on the disease in Porto Rico were recorded in a circular (1)[1] and a note in Phytopathology (2). The following is part of the text of the note:

"In February, 1920, gumming of sugar cane was identified by the writer in two car loads of cane from the Trujillo Alto district, Porto Rico. The gummy exudation, varying in color from a lemon-yellow to that of honey, at the cut ends of the cane stalk, together with the bright-red vascular bundles in cane in the more advanced stages of the disease, are symptoms that agree with the gumming

[1] Numbers in parenthesis refer to references listed at the end of this paper.

of cane of Australia as described by Cobb and studied by E. F. Smith. Cultures of gum were made on potato agar, and the bacterial colonies as well as the transfers of these to potato plugs agree with those of *Bacterium vascularum* described by the last author. Masses of the bacterium in pure culture were smeared on needle-pricked surfaces of leaves of ten healthy, young "Otaheite" canes in an isolated greenhouse. After three months from inoculation all these plants showed, when cut, typical gummy exudation from the fibro-vascular bundles.

"Immediately upon the recognition of the fact that gumming disease exists unmistakably in Porto Rico, a special survey was made of all the principal sugar-cane growing centers, and it was found that the disease is not wide-spread and that the exact locations of its occurrence are only in the Trujillo Alto district in an area ofabout fifteen kilometers. Specimens of the diseased cane were shown to men who have planted cane for years but all were unanimous in declaring it a new thing. One planter stated that he saw it to a very limited extent the previous year. The disease occurs in places where no new introductions of seed were made for many years past, and it was found in the oldest varieties grown on the Island, namely, "*Otaheite,*" "*Rayada,*" "*Crystalina*" and "*Cavengerie*" or "*Colorada.*" The "Otaheite" is the most severely infected, while the "Cavengerie" is the least infected variety. It is very likely that the disease has been here in insignificant proportions for some time in the past, but the unusually heavy rains at the beginning of the present year have most likely aided it in taking on the form of an epidemic in one district where "Otaheite" is still generally grown. Whether the disease will subside with the advent of normal rainfall remains to be seen. The only safe way to check it is through the planting of resistant varieties. So far the disease has not been observed in the variety "*Yellow Caledonia.*" The fact that "*Rayada* is susceptible makes it rather a difficult problem here, as this variety is very largely grown all over the Island. "Cavengerie" shows decided resistance but it is not a very desirable variety here from the millers' point of view."

The following year, beginning January 1921, the disease was found to have spread to a much larger area. Cane in the vicinity of Caguas, Cidra, and Cayey were found heavily infected with gumming. From Morovis and Barros, in the interior of the Island, heavily gum-diseased canes were repeatedly received. The disease was also found at Corozal, Aibonito and Barranquitas. During that year gumming disease was found to have spread at least 25 kilometers in westwardly and southwardly directions in advance over the infected area of the previous year.

At the beginning of the third season, the 1922 harvest, the disease was located in the interior as far as Adjuntas; south and southwest it has advanced to Santa Isabel, Peñuelas, Guayanilla, and Yauco; on the north coast it has advanced from Bayamón towards Vega Baja; and at the eastern end of the Island it was found in the vicinities of Humacao, Las Piedras, Fajardo, Río Grande and Carolina. In the previously infected zones around Trujillo Alto,

Río Piedras, Bayamón and Cayey the disease became more common in a larger number of properties, and it became noticeable in the more resistant cane varieties as well.

### THE OCCURRENCE OF THE DISEASE OUTSIDE OF PORTO RICO.

Contemporaneous with the outbreak of gumming in Porto Rico the same disease was, and is at the present writing, existing in Australia. There exist records of the occurrence of the disease in other countries and these records are summarized by Dr. E. F. Smith in his treatise "Bacteria in Relation to Plant Diseases" published in 1914. In summarizing the geographical distribution of the disease he states as follows:

"This disease occurs in New South Wales (Cobb, Greig Smith), Queensland (Tryon, Cobb), Fiji Islands (Clark), Mauritius (Boname), Java (Went), Borneo (Kruger), New Guinea (Cobb), and Brazil (Dranert). From various statements in Spegazzini's paper on Polvillo or humid gangreen of the cane I believe this disease is also present in Argentina, Tucuman province. It is probable not in North America. No complaints have been received from the cane fields of Louisiana. Dr. Went did not find, or hear of it, in his tour of inspection in the Dutch West Indies in 1902. It has not been reported from the British West Indies nor from Porto Rico. The writer did not see it, or hear of it, in Cuba in 1904, nor was it seen in 1907 by John R. Johnston, Assistant in the Laboratory of the Plant Pathology, who spent eight months in the West Indies, visiting Cuba, Jamaica, Trinidad, Guiana, Venezuela, Barbados, and Porto Rico, and who was instructed to look for it especially. Recently a top rot of sugar cane has been reported from Cuba by Cook and Horne, but their account leaves very much to be desired. It does not occur in the Sandwich Islands (Cobb). Sereh and cane gomosis are said by Dr. E. J. Butler to be 'unknown or rare' in India (letter to our Secretary of Agriculture, April 21, 1903). It is a disease most prevalent in the Southern Hemisphere but one likely to occur wherever cane is grown. The Java top rot as described and figured by Wakker is not this disease. Possibly, however, it may have been confused with the Java heart-rot or with the pokkabong."

The earliest recorded date of the occurrence of the disease is that of 1869, when F. M. Dranert (3) reported the disease from Bahia, Brazil, and he stated that the disease existed there to an alarming extent for at least six years previous to that date.

In 1894 it was reported from Pernambuco, Brazil, that the disease was greatly diminished by the substitution of resistant cane varieties among which there is mentioned a "dark claret-colored cane with a still darker stripe, called here 'Louzier.'" This cane is probably our *caña* Colorada or Cavangerie. In the same year M. Boname reported the disease from Mauritius.

One year back, in 1893, Cobb reported the disease from Australia (New South Wales) and brought forth the idea that it is caused by a bacterium. Two years later Tryon reported on the gumming disease of sugar cane in Queensland.

In 1902 Greig-Smith published an account on the reactions of the bacterium of gummosis which he isolated from diseased cane and cultured in pure state.

In 1904 Dr. E. F. Smith published a paper on his inoculation experiments with a pure culture of *Bacterium vascularum* which he obtained from diseased cane from Australia, establishing the causative relation of this organism to gumming disease of cane and he attempted to show that acidity of the cane is a factor in immunity to this disease.

De Gomziekte van het suikerriet (the gum disease of sugar cane) of Java has been discussed in publications by J. Groenewege (4) in 1914 and again in 1916 (5). The same disease has later been studied by Miss. G. Wilbrink and the results and observations of this author were published in 1920 (6).

In the last three publications from Java the identity of the Javan gum disease with the one described from Australia is amply discussed. Groenewege maintains that the gum disease of Java is identical with the gumming of Australia. Wilbrink does not share that view and maintains that the identity of the two diseases could be established only if they were observed together in one locality. In reality there exists a marked symptom by which the two diseases are distinguished from each other, namely, the exudation of a copious yellow gummy mass which is the most characteristic symptom in the Australian and Porto Rican gumming disease and which is lacking entirely in the disease of the same name at Java. All of the investigators in Java agree that there is no exudate from affected canes. It is true, the extent of gum flow in quantity from the cut ends of the disease cane may become influenced by atmospheric conditions to a certain degree, but it is always in evidence no matter how small the quantity, under a rather wide range of climatic conditions such as at Brazil, Australia, Mauritius, Porto Rico and to say the least in the greenhouses of Washington where Dr. Smith has produced the disease, exhibiting the symptom of gummy exudation, with pure cultures of the organism isolated from cane which became diseased in Australia. It is therefore difficult to accept the view that the same organism

which produces the characteristic gum flow in several countries however much they differ from each other climatologically, should lose entirely that very pronounced character in Java. It seems that the gum disease of Java is caused by another organism.

It would be incorrect to suppose that the gum flow in diseased cane is not a primary or direct symptom of the disease but is due to other organisms which may follow the initial entrance of *B. vascularum,* because in the first place cultures of small drops of the gum itself have, in many instances, produced uncontaminated growth of *B. vascularum* unassociated by any other organism, and secondly, pure cultures of *B. vascularum* on a 2 per cent glucose, 5 per cent peptone, almost neutral agar medium are exactly like the gum; exuded from diseased cane, in color, consistency and copiousness. It is evident, therefore, that the gummy exudation is a natural character of *B. vascularum* and is present whether the organism lives in the sugar cane or in an artificial food medium in the laboratory. This same phenomenon should occur in Java, provided *B. vascularum* is the organism associated with gum disease there. It should be stated here that in using the word "gum" in connection with this disease it does not imply a true gum but only in the sense of a sticky substance, because in reality it is not more than a yellowish zooglia mass of bacteria each individual bacterium possessing a gelatinous and sticky outer covering. One becomes readily convinced of it when a drop of newly exuded gum of a freshly cut cane is examined under the microscope, then an ordinary mass of bacteria is seen and nothing else. If the exudate is somewhat dry on account of exposure to dry air the individual bacteria are less distinct as they do not readily separate themselves after they are cemented together through drying and in this condition it may resemble a gum. In free-hand sections of cane affected with gumming the individual bacteria are easily detected in some of the vessels of the fibers. (Fig. A.) There occurs, sometimes, on account of injury, a homogenous gum-like substance, variously colored, in the vessels of sugar-cane fibers, but this substance does not contain normally any detectable masses of bacteria.

Regarding the relation of Sereh disease to gumming it is quite clear that not only is our gumming distinct from Sereh, but also the Javan gum disease has nothing in common with Sereh according to the later investigation of Wilbrink.

ECONOMIC CONSIDERATIONS.

Gumming of sugar cane is a serious disease in view of the fact that it produces decay of the growing points of young as well as mature canes, naturally where young canes are attacked they stop growing and ultimately die. The disease prevents the development

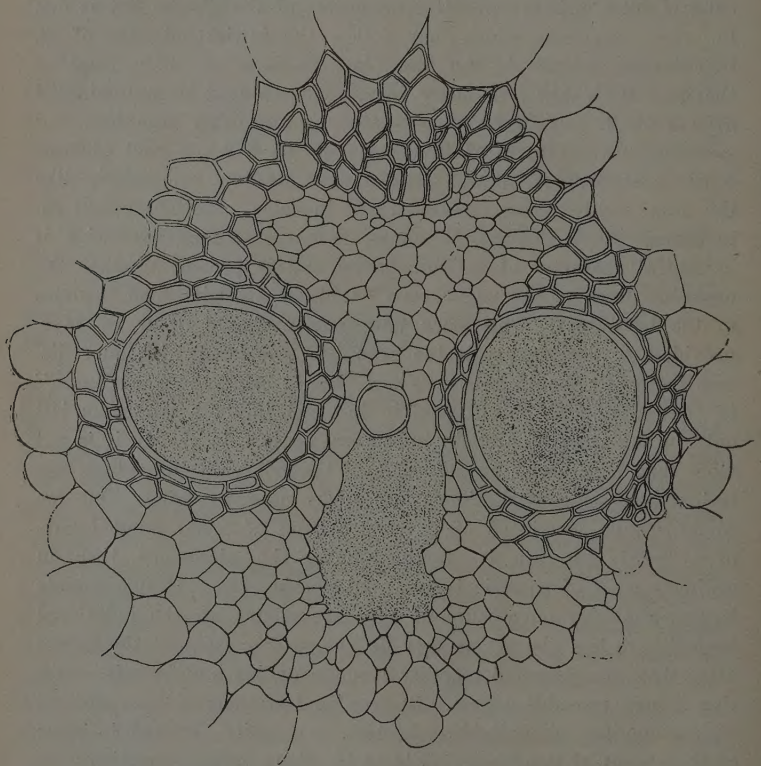

FIG. A.—Cross section of a fiber of sugar cane affected with gumming disease, showing the masses of bacteria in the vessels.

of the second-generation canes in the ratóons, and the presence of the gum causes difficulty in the elaboration of sugar at the mill—it interferes with cristalization in the vacuum pans or tachos. If gumming disease attacked with equal severity all varieties of cane, the sugar-cane industry would be destroyed in a few years. For-

tunately there are cane varieties that are not affected by it and some only in a slight degree. The most susceptible varieties are at the present time the least cultivated ones in Porto Rico, except in one locality around Guayanilla where the susceptible Otaheite cane is still predominant. The most commonly grown canes here in Porto Rico are the varieties Cristalina and Rayada, and these exhibit a certain degree of resistance to the disease, yet they are quite commonly found diseased in the infected zones, especially if they are planted together with infected Otaheite canes. The apparent resistance of certain cane varieties to this disease is not thoroughly understood, it may be an inherent quality such as the reaction of the cell contents to the invading organism, structural differences which may obstruct its entrance, or the absence or scarcity of carrying agents which may frequent in their visits some varieties more than others, but whichever factor is responsible for the greater resistance of, for instance, Yellow Caledonia to the disease at the present time, nevertheless it is possible for a change to take place in the requirements for the development and the adaptability of the bacterium and thus enabling it to attack hitherto resistant canes. As long as there exists a source of infection any cane variety might lose its resistance and adapt itself to the parasite.

Besides the immediate destruction of the tops of growing cane which results in the death of younger canes and a reduction in tonnage, gumming causes a rapid decay of canes after they are harvested. In this way mature canes may lose a large proportion of their sucrose if they are to remain in the piles or in the loading cars for several days before being ground.

It is impossible to give an estimate of the loss caused by the disease because it would be necessary first to determine the quantity of Otaheite canes present in the fields, since this cane is the one that suffers more materially of this disease. In the largest number of fields where this disease occurs there exists a mixture of varieties, in various proportions, usually Cavengerie, Caledonia, Cristalina, Rayada, and Otaheite with its close relative the Calancana. The last two varieties are the most susceptible ones to gumming; next follow Cristalina and Rayada, then Cavangerie is only slightly infected and Caledonia is practically immune so far. The amount of loss in any plantation, due to gumming, would depend therefore primarily on the nature of the predominating variety planted.

Some growers have expressed the view that gumming disease is less severe in ratoon than in plant cane. This is true at first glance

because the infected early sprouts die soon on account of the disease, that leaves the stool apparently free from the disease, though with fewer canes, for a time at least; but the bacteria have since found their way into the older shoots, and before long some of them will show symptoms of the disease and dead tops, and thus the stools become thinned out, and lastly when the crop is harvested those few apparently full-grown and mature stalks will show the gummy exudation from their fibers when cut.

FIG. 1.—Gumming of sugar cane, gum drops exuding from cut surface of cane.

In one ratoon field the disease actually eliminated itself with the disappearance of the few stools of infected Otaheite in amongst the resistant Cavangerie and Yellow Caledonia.

### THE SYMPTOMS OF THE DISEASE.

The principal symptom of the disease is the yellow gummy exudation from the cut ends of affected cane, and it is so striking that few can fail to become aware of its presence. (Fig. 1.) The exudate varies somewhat in color and abundance. At times it is grayish yellow and somewhat watery but more often it is lemon yellow and thickly gummy. In almost every case, and especially where the disease was present in any marked quantity of the harvested cane, the mill men recognized the disease by this symptom before their attention was called to it. This helped to ascertain the distribution of the disease.

## GUMMING DISEASE OF SUGAR CANE

Symptoms of inner leaf showing reddish specks on light green areas near margins of leaf.

Diseased leaf showing long, dark brown streaks.

At first it was thought that gum-diseased cane could be recognized only by the yellow gummy exudation from the cut ends of the cane, but further observation showed that the disease can be located in growing cane, before they are cut, by a peculiar appearance in the leaves. The leaves, and mostly the younger and innermost, not fully unrolled ones, show, in the early stages of the gum disease, pale green to almost pure white patches and (Fig. 2) longitudinal bands or streaks. These light-colored areas become often sprinkled with dark-red small spots or narrow and short streaks. (Fig. 3.) Such leaf symptoms can be found in young shoots or in older ones in the not quite unfolded basal parts of their inner leaves. In the outer maturer leaves long dark brown streaks may be found. (Fig. 4.) In older cane and where the disease is more advanced the inner leaves possess long, sometimes lighter and sometimes darker gray, dead, stripes usually about 1 centimeter in width. These stripes are usually found towards the middle of the leaf blade. This feature distinguishes this symptom from the ordinary drying of leaves which occurs in cane either because of white-grub injury, borers or drought. In the latter cases the edges of the leaf commence to dry first. In gum-diseased cane the dry stripes are usually in the interior of the leaf, while the edges may remain green for a long time. This phenomenon is due primarily to the partial infection of some of the fibers; naturally only the cells surrounding the infected fibers die first and result in the dead-stripe appearance in the leaf. Usually the tops of gum-diseased cane showing the dry stripes will not be as widely unfolded as in healthy cane, the dead longitudinal areas or stripes in the leaves preventing the straightening out of the leaf blades, therefore the tops in gummy cane usually stand up erect and are more or less

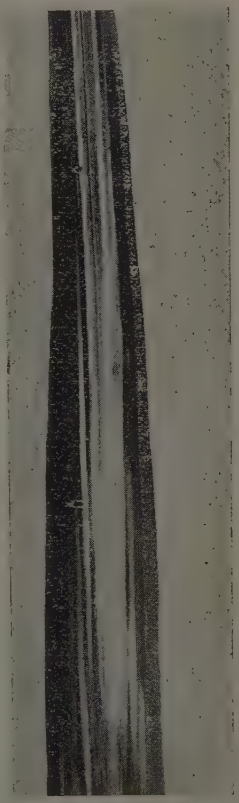

FIG. 2. — Light - green areas in first stages of gumming disease in the leaf of a young sugar-cane plant.

unfolded. In the latter stages of the disease an odorless decay sets in the tissues of the growing points of the cane. At this stage the outward symptoms bear resemblance to the top-rot condition of cane

FIG. 3.—Reddish to dark-brown spots and narrow and short streaks on light-colored areas at the base of a sugar-cane leaf of a gum-diseased plant.

FIG. 4. — Long dark-brown streaks in outer leaf of gumming-diseased sugar-cane plant.

caused by borers and *Plasmodiophora* disease. In the last cases the cause of top rot is due entirely to the interference with the normal functioning of the fibrovascular system. However, whether it is a bacterium as in gum

disease or a *Plasmodiophora* as in dry top rot which fills up the water-conducting vessels, or whether it is a mechanical cut made by an insect, thus breaking the connection between the roots and top of growing cane, the effect on the growing point where the new leaves issue from is the same. In gumming disease, in addition to a clogging of the fibers, there is a direct decay of the tender tissues of the top caused by this bacterium as is evidenced by the red coloration of the tissue between the fibers.

Another phenomenon found with gum disease is the red coloration of some of the fibers themselves in severely affected cane. This is not a primary symptom of gum disease, but it indicates that the phloem in some fibers, or in these which show the red color, have died. Canes which show exudation of gum from a majority of their fibers do not in many cases have a single red fiber. In many instances a severe stunting of the stalks and the presence of grayish longitudinal depressions along the internodes was quite common. In gumming top rot there may be present masses of gum between the leaf sheaths and the stalk.

In summarizing the symptoms of gum disease as it occurs in Porto Rico we must distinguish between the primary and secondary symptoms. The primary symptoms, or those which are always associated with gum disease, are the yellow gum exuding from the fibers of cut canes, the light areas sprinkled with dark-red little streaks in the younger portion of leaves, brown long streaks, and light to dark gray, more or less wide dead stripes in the older leaves, and top rot. Red fibers, and even stunting of cane may or may not occur in gum-diseased cane, depending on the severity of the infection. While the gum-flow symptoms is the easier to detect after the cane is cut, the leaf symptoms are more important, because by these it is possible to detect the disease in the field before the cane is cut, and it should be taken advantage of in controlling the disease. Wherever possible, diseased stools should be cut after the healthy stools have been harvested. This is hardly possible in severely infected fields, but it should be borne in mind that infected *machetes* can introduce the disease into healthy cane.

### THE CAUSE AND BEHAVIOR OF GUMMING DISEASE.

As stated in the introduction, *Bacterium vascularum* was isolated from diseased cane and it was proved by inoculation trials that this organism is the cause of the disease in Porto Rico. However,

some difficulty was experienced in the first attempts at isolations. For a time no growth was produced in agar plates from diluted gum, but it was soon discovered that the agar medium used was of a too strongly acid reaction. When the work was repeated, using sucrose or glucose agar titrated to only + 1 or + 2 Fuller scale and sometimes leaving the agar at about neutral, copious growth of the bacterium, from diluted gum, was obtained in three or four days. In thick sowings growths was visible to the naked eye the following day.

From the sudden appearance of gumming disease here in the fields, where no new seed introductions were made for some time and in varieties which have been grown here for many years, no definite opinion could be formed as to its future possible spread, but now it is certain that it is spreading rapidly and that it is becoming a general epidemic. In attacking this problem at the Insular Experiment Station it was felt that the first thing to know is the manner by which this disease is spread. It became evident from the inoculation experiments that it could be transmitted by contact. Could the disease be transferred to healthy seed planted in previously infected soils? This was the second problem which was deemed important to solve. Gum-diseased cane pieces, the buds of which were removed, were split and these were tied to healthy seed and planted in new soil. Twenty seed of each of the varieties Caña Colorada, Yellow Caledonia, Rayada, Otaheite and P. R. 260 were used in the trial, each variety being planted in a separate row. In addition five seed of each of the above varieties were planted in the same rows but alone without infected cane. Practically all the healthy seed in this whole planting germinated and no sign of disease was noticeable in the young plants in spite of the fact that at first their buds and later their roots had been in contact with gum diseased cane pieces which were gradually decaying in the soil. They all made a good normal growth, and when the whole plot was harvested at the age of eight months, there were no trace of gumming in the stalks of any of the varieties used. The ratoons of these canes sprouted normally and no disease symptoms were noticeable in them when they were cut 10 months later. Apparently the soil is not the proper means through which infection might be carried to the roots of healthy seed. In another experiment diseased seed were planted with the view to allow those to sprout, as only about 25 per cent germinated; healthy Otaheite seed were planted in the holes of the ungerminated seed. There

was no gum disease produced in the replants of the susceptible Otaheite in this manner. However, when *Bacterium vascularum,* isolated from diseased cane, was introduced with a needle into the young leaf spindle of Otaheite and Rayada canes growing in the field the disease was reproduced with all its symptoms. That indicates that the air route is the path of transmission for this disease.

Stools of cane which contained several diseased stalks were dug up and all the stalks and shoots cut back. The stubble with their roots were transplanted amongst young healthy canes in an isolated field. It was noted that some young shoots which came up from those diseased stools showed at the very beginning the symptoms of gum disease, and what is more significant the disease was later found in the adjacent healthy stools as well. It is therefore evident that the infected ratoon left in the field constitutes a positive source of infection and that the disease can be carried over from these to the young canes of healthy stools. The possibilities are, therefore, that the disease can be transferred to growing cane by insects, by the cutting instruments and by driving rains, but by eliminating the diseased ratoons the primary source of infection is destroyed, since the soil does not form a favorable abode for the bacterium. Artificial inoculations in the roots of susceptible canes gave negative results. This can not be explained on the ground that the acidity of the soil does not favor growth in the bacterium.

A test was made with liming soils and then inoculating the soils with a pure culture of the organism, but the canes which grew in these soils did not become diseased. It is very likely that soil infection does not take place not so much on account of an acid condition but because the roots of the cane do not constitute a receptive organ for the entrance of the bacterium. From field observations and from experimental tests it was learned that the growing tenderer tissues of the top leaves and points of the stalk are the most susceptible parts of the cane plant to the disease. Even resistant cane varieties may become infected when young, by artificial inoculation through the top leaf spindle. This was noted in the varieties B–208, D–625, and G. C. 606.

The course of the development of the disease from diseased seed begins with the sprouting buds. The bacterium which is present in the fibers of the diseased seed passes on directly into the unfolding bud, as some fibers of the seed are directly connected with those of the bud. If the bacterium has reached the interior of the bud before it started to swell, that bud will most likely not germinate.

On the other hand, if the bud is not contaminated, and swells rapidly, and the seed piece begins to decay early, the shoots from this bud may grow healthy, as the bacterium may become arrested in its growth on account of the acid condition of the seed tissues. Thus in planting diseased cane seed a considerable number of them do not germinate, some produce healthy shoots and some diseased shoots. The diseased shoots show the symptoms of gumming in their leaflets, usually the disease progresses until the shoot is stunted and killed; soon later another shoot, more advanced in growth, begins to show the disease, apparently a secondary infection, and so the younger infected shoots die in their turn but perpetuate the infection from one to another until good-sized canes are reached in which the disease is left to stay in the cane, as these do not die readily on account of the disease. The organism lives in the fibers but does not break through into the parenchyma cells of the maturing cane stalk, except in the top where it is able to enter the parenchyma cells and causes these to decay. In the same stool there may be diseased and healthy stalks. If the diseased stalks are removed the stool may remain healthy.

In the ratoons the disease is propagated to a large extent by the cutting instrument. When the canes are cut the very young shoots in the stools are also cut away. The cutting instrument which may have gum on its blade introduces it into the tender inrolled leaflets of the healthy younger shoots and these become diseased.

The infection has been observed to have been carried from one field to another through considerable distances.

### VARIETAL RESISTANCE.

One of the most striking features of this disease is its preference for certain varieties of cane. This fact has been noted years ago at Pernambuco, Brazil, and though little has been spoken of it of late in reports on the disease from Australia, it is very accentuated here. Of our four or five common varieties one, Otaheite, is very susceptible and another, Yellow Caledonia, is practically immune. Experiments to test the resistance of varieties had to be done by artificially inoculating them with a pure culture of *Bacterium vascularum*. This method is not quite satisfactory since it does not represent a truly natural state of affairs, but it shows the possible susceptibility of each variety tested.

The first experiment on susceptibility of varieties was made by inoculating a number of young canes of B–208, D–625, Rayada, and

Otaheite. The expanded portions of the whorls of leaves at the top were cut off and *Bacterium vascularum* masses were smeared on the cut adges of the remainder of the whorls, that is, on the lower more or less inrolled portions of the leaves in the cane top, and these were covered with paper bags to prevent too rapid drying out. At the end of four weeks the typical red-brown spotting and streaking and drying of the top leaves were noted in the varieties Rayada and Otaheite, in B–208 the disease symptoms developed to a lesser degree and in D–625 the symptoms were faintest. Decay set in in the tops of the infected Rayada and Otaheite stalks and they finally died while B–208 remainded stunted and D–625 seemed to have recovered.

During the year 1920 a larger variety test was made at the Insular Experiment Station to find which of our best canes are resistant to the disease. The results thus far are very encouraging because many of our good canes are resistant to artificial inoculation.

The following is a list of the cane varieties which were tried out and their reactions to the disease as noted at the end of the first crop:

GROUP I.

STRONGLY SUSCEPTIBLE.

| | | | |
|---|---|---|---|
| Calancana | Rayada | B–376 | P. R.–491 |
| Otaheite | Cristalina | P. R.–358 | P. R.–487 |

GROUP II.

SLIGHTLY SUSCEPTIBLE.

| | | | |
|---|---|---|---|
| P. R.–260 | B–3405 | P. R.–207 | P. R.–328 |
| Colorada | Sealy Seedling | | |

GROUP III.

SUSCEPTIBLE IN YOUNG STAGE ONLY.

| | | |
|---|---|---|
| B–208 | D–625 | G. C.–606 |

GROUP IV.

RESISTANT OR IMMUNE.

| | | | |
|---|---|---|---|
| Uba | D–109 | B. H.–10(12) | P. R.–417 |
| D–448 | B–109 | P. R.–333 | P. R.–334 |
| Yellow Caledonia | P. R.–308 | P. R.–229. | P. R.–370 |
| B–3412 | P. R.–209 | P. R.–292. | P. R.–272 |
| D–117 | P. R.–202 | P. R.–318 | B–1809 |
| B–6292 | P. R.–230 | P. R.–309 | |
| D–433 | B–347 | P. R.–219 | |

## CONCLUSION.

The disease is easily transmitted by any conductor or carrier such as cutting instruments, biting insects, etc., into the growing portions of the leaves and young stalks of susceptible canes. The soil, however, is not the place through which infection can take place. Healthy seed may be planted in the same holes from where diseased canes were pulled out. In this respect it is similar to the "Matizado" or mosaic disease. Of the common canes the Caña Blanca (Otaheite, Burbon, Lahaina) and Calancana are the most susceptible canes we know, and as regards gum disease these are in the same relations to the other sugar canes as the rat is to men in the case of bubonic plague. It is quite certain that gumming disease was not present at Guayanilla before this year but it got in there because most of their fields are planted to Otaheite. This cane is grown in the interior to some extent and the disease has simply followed this cane, wherever it happened to be. At the Insular Experiment Station this same cane, in amongst a large number of other varieties, was found infected.

Ever since 1920 we have insisted on the extirpation of the disease and the avoiding of the use of Otaheite cane as seed. In the Trujillo district D–109 has been planted with success. Very drastic measures will have to be taken on the south coast to eliminate Otaheite plantings and destruction of the ratoons of some to prevent the disease from getting into the Cristalina and Rayada varieties, which are susceptible to the disease. In our experiments Rayada and Cristalina have shown to be susceptible to the disease, but in looking over cars of cut cane from infected zones it became evident that the last two varieties possess resistant properties, and it is because of their being planted together with the highly susceptible Otaheite that they contract the disease from it.

When gumming disease was first found here just two years ago, we at once advised the planting of healthy seeds and varieties which are resistant to it, but we found later on that the greatest source of infection is the diseased ratoon which is left in the field, therefore the diseased stubble must be removed in order to prevent the perpetuation of the disease in the old infected areas. But it would be impossible to detect diseased from healthy stubble, especially in fields where there is not more than 2 or 3 per cent infection if there were no means by which to recognize the diseased cane before their being cut. For this the leaf symptoms should be looked

for at the time of harvesting cane, and such stools as show the disease in the leaves should be left uncut. This practice will serve a double purpose; first, it will thus leave diseased stools marked to be removed afterwards, and secondly, it will avoid to a large degree the possibility of carrying the disease from infected to healthy stools by the cutting instrument.

During the first seasons only a few scattered samples of gummy Rayada, Cristalina, and more rarely Cavangerie or Colorada were seen. All of the outbreaks of the disease were first in Caña Blanca and in the recent cases of Coamo and Santa Isabel the new infection could be traced directly to infected Caña Blanca seed which were brought there from infected zones. It is therefore evident this cane is not only deemed to become totally infected but that it also acts as a carrier of the disease to the more resistant Rayada and Cristalina. On the other hand, the red cane (Cavengerie) and Caledonia should be propagated on a larger scale, at least for the present, as these varieties on account of their resistance will tend to check the march of the disease. Another red cane known as "D–109" has shown so far a remarkable resistance to the disease and it is advisable to procure seed of this kind and replace the white Otaheite in the hill lands.

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# RECENT DEVELOPMENTS IN THE STUDY OF THE NATURE OF MOSAIC DISEASE OF SUGAR CANE AND OTHER PLANTS.

By JULIUS MATZ.

Mosaic diseases of plants, including mosaic of sugar cane, have of recent years come to be generally considered as infectious and obviously caused by microorganisms which were supposed to be ultramicroscopic and invisible even with the aid of the usual staining materials. Most investigators have apparently relied in the past, in the studies of mosaic diseases, on the culture method to detect the existence of organisms; having failed in that and not recognizing any familiar forms of bacteria or fungi in the host tissues the problem was left in the field of inexplicable phenomena. More recently, however, within the last three years, new light was thrown on the subject, and it seems that the causes of mosaic diseases are in a fair way to become clear. The new procedure in the investigation is centered along cytological lines and is based on comparative studies on the contents of cells from diseased plants and contents of cells from similar but healthy plants.

In 1919 the writer started histological studies of sugar-cane mosaic and it was soon found that in advanced stages of the disease small portions in the interior of mosaic cane stalks become slightly bleached, as well as light brown and dark brown. In these discolored or darkly colored tissues the cells are filled with a more or less hardened, or compact, densely but finely granulated, often slightly browned plasma.

Since the publication of the above facts in an article entitled "Infection and Nature of the Yellow Stripe Disease of Cane (Mosaic, Mottling, Etc.)" in the JOURNAL OF THE DEPARTMENT OF AGRICULTURE OF PORTO RICO, Vol. III, No. 4, Oct. 1919, two investigators, namely, Dr. Kunkel, writing in 1921 on corn mosaic in Hawaii (1), and, more recently Dr. Palm, in his Bulletin treating on the cause of tobacco mosaic (2), refered to my findings in sugar-cane mosaic diseased tissues as something similar to what the first author found in corn mosaic and the second author in tobacco mosaic. Kunkel stated that in addition to the larger bodies which he found in the leaves of corn mosaic, he is "able to confirm the observations of Matz as regards the occurrence in diseased cane

tissue of cells filled with a hardened, granular slightly brownish substance." He further states that "Cells filled with exactly the same kind of granular material are also present in the stalk tissues of corn suffering from mosaic. * * * What this substance may be or what relation it may have to either the cane or the corn disease the writer is unable to suggest."

He published some very carefully executed drawings (plate 5, figs. A to M.) of what he calls intracellular bodies and which he considers as foreign bodies believed to be a living organism in the diseased cells of mosaic corn plants. These bodies are shown attached in various positions to the nucleus of the host cells.

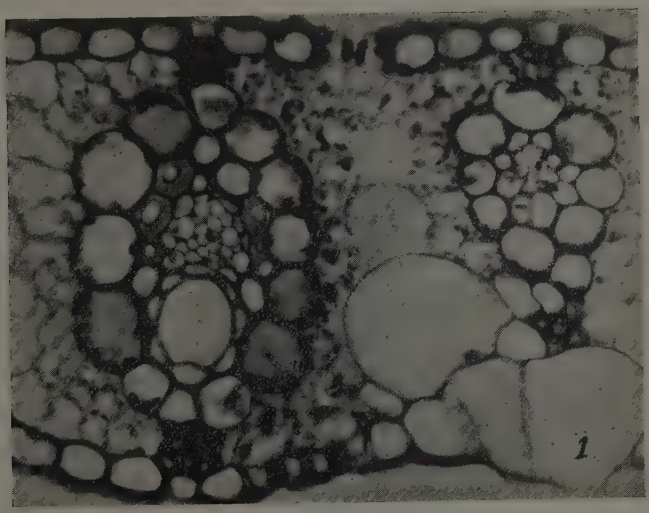


**FIG. 1.—Section through a healthy sugar-cane leaf, showing chloroplasts in cells.**

Iwanowski in his studies of tobacco mosaic published in 1903 (3) illustrates (in Table II, fig. 8) strikingly similar bodies, to those shown by Kunkel, in identical positions attached to the nuclei of the host cells, but Iwanowski disregards these as a possible cause of the mosaic disease on the grounds that these bodies are altogether too large for the fine pores of filters to pass through. The infectious principle of tobacco mosaic is known to filter through fine-porous filters. This investigator considers these larger bodies as abnormalities in the cytoplasm of the cells due to mosaic infec-

tion, but mosaic itself he maintains to be caused by a minute bacterium which he differentiates from the other cell contents by using Loeflers methelene blue and eosin, the chloroplast and protoplasm taking the red stain while the nuclei and the bacteria take the blue stain.

The writer has applied these stains to sugar-cane mosaic leaf tissue and the two colors differentiated the chloroplasts from the nuclei but no blue bacteria were detected. However, the behavior of tobacco mosaic is not like the sugar-cane mosaic, as will be pointed out later, and it is possible that the "bacteria" are not localized in the same elements in the two host plants' tissues.

Palm in his very recent work with tobacco (2) states: "In a large number of cells of the mosaic-diseased tissues it was possible to observe the occurrence of foreign elements, viz., of fairly large, more or less peculiarly shaped corpuscles, or very small granules of varying size." He adds in a note: "Matz (1919) has found such corpuscles in mosaic-diseased sugar cane in West Indie, and Kunkel (1921) in mosaic-diseased maize in Hawaii. * * *" He further states: "In the cells with the above-described larger corpuscles or in others where they apparently did not occur, it was possible, especially in the later stages of the disease as already mentioned, to find a second foreign cell element, consisting of extraordinarily small granules."

"These granules * * * occur in the cells in larger or smaller numbers. They frequently lie in irregularly shaped conglomerates in the cell plasm sometimes the cell lumen being completely filled." This point agrees very well with my observations on sugar-cane mosaic.

In my article above refered to I stated that the granular substance in the cane mosaic is made up of a mass of small hyaline granules, more or less uniform in size, but that their exact size and form could not be ascertained, due to the fact that the whole mass is in the form of a compact plasma, that the granules are smaller in size than ordinary bacteria and appear like nuclear granules in a mass of cytoplasm. This characteristic granular plasma was also found in cells of mosaic leaf sheaths of cane.

While Dr. Kunkel lays more emphasis on the larger bodies in the cells of corn mosaic and is not able to define with certainty the relation of the minute foreign granules in either the corn or cane mosaic, Dr. Palm, however, finds it convenient to compare these minute granules of Iwanowski, Matz and Kunkel to the gran-

ules found as a consequence of variola infection and other virus diseases in human beings and animals. From this he draws the conclusions that mosaic disease belongs to the chlamydozoonoses; that the different forms of granules found in mosaic-diseased plants are either different stages of *Strongyloplasma* or by-products of this organism, and he names the organism which causes the mosaic of tobacco *Strongyloplasma iwanowskii,* giving the honor to Iwanowski who twenty years ago maintained that he saw bacteria in tobacco mosaic-diseased plants. Although Dr. Palm states that the organism which is found in tobacco is similar to the substance which

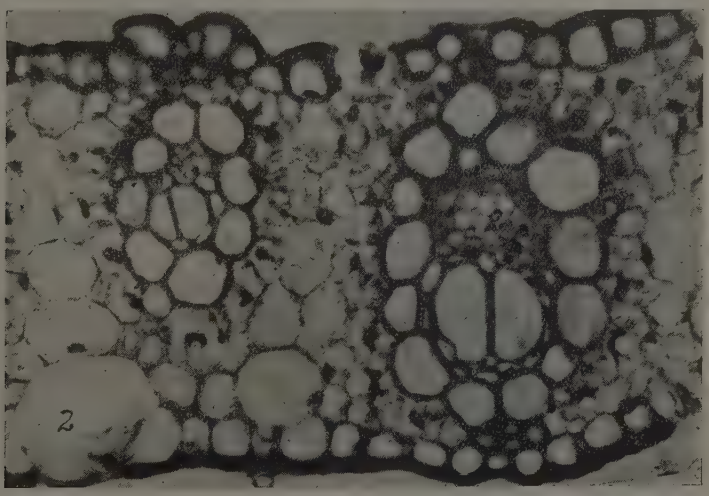


**FIG. 2.—Cross section through mosaic leaf of sugar cane, showing destruction of chloroplasts.**

I found here in sugar cane, we do not necessarily have to accept *S. iwanowskii* as the name for the plasma found with the sugar-cane mosaic disease in view of the fact that the two diseases do not behave in a similar manner. Tobacco mosaic is transmitted by contact while sugar-cane mosaic is transmitted by special carriers or special methods only. The infection of tobacco mosaic may take place through diseased plant residue in the soil, while such is not the case with the sugar-cane mosaic. Perhaps the most important, distinction between the two diseases is their apparent specific characters. Tobacco mosaic is not known to have been transmitted in the field to cane from the tobacco, nor is it known that cane mosaic

is transmitting itself to tobacco. There exist extensive tobacco plantations free from mosaic in the Cayey District where cane is heavily diseased with mosaic. Evidently the organisms which cause mosaics in plants are of a groupe which have common general or generic characters, but there must exist specific differences between these since they exhibit marked differences in their behavior, and the sugar-cane plasma if it is to have a name it will have to have one of its own. Further work was continued at the Insular Experiment Station on the same phase of this problem and new and interesting facts have come to light that may be stated briefly in the following.

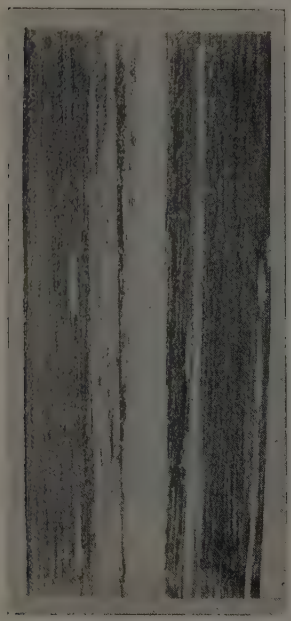

FIG. 3.—Early stages in mosaic diseases of sugar-cane leaf.

Of the numerous methods employed in studying plant tissues, Jeffreys' formula of corrosive sublimate and picric acid, with the addition of 5 c.c. glacial acetic acid, was found convenient to use. Healthy and diseased portions of sugar-cane leaf were killed and fixed in the above mixture for two or three hours, passed through several washings in 70 per cent, 85 per cent and 95 per cent alcohol, then the material was transferred to butyl alcohol and left there until it lost all its green color, then it was placed in melted paraffin and embedded.

Sections of cane-leaf tissue were stained with iron-alum haematoxylin, orange G, and methelene blue and eosin. One feature was very pronounced throughout, namely, the very evident destruction of chloroplasts in the diseased portions of the leaf tissue. Figures 1 and 2 are photographs of a healthy and diseased leaf portions respectively. The two leaves were as nearly alike in size and age as could be estimated. In the healthy leaf the larger cells surrounding the fibro vascular bundles contain many chloroplasts; the parenchyma between the bundles are also filled with numerous chloroplasts, while in the diseased tissue the chloroplasts are few and are evidently mis-

shaped and broken up. In stained sections they look like mere ink spots, one or two in a cell. This destruction of chlorolpasts is a symptom of sugar-cane mosaic and it fixes the seat of the disease more definitely. Apparently the cell walls and other cell contents are not affected, but the chloroplasts are gradually destroyed.

Sections of single discolored stripes of a leaf in an early stage of infection were made (Fig. 3), and it was seen that the breaking up of the chloroplasts begins with a reduction in their size. The chloroplasts in the healthy or green parts of the same leaf were normal in their size and numbers, while in the discolored or pale-green stripes chloroplasts in all stages of reduction were noted.

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# DRY TOP ROT OF SUGAR CANE.

## A VASCULAR DISEASE.

By JULIUS MATZ.

Since the latter part of the year 1919 the writer has had under observation a fibro-vascular disease, which is manifested outwardly by a dry top decay, of the sugar cane (*Saccharum officinarum* Linn.) in Porto Rico. A description of the organism *Plasmodiophora vascularum* n. sp. associated with the occurrence of this disease, was published by the writer some time ago (1).[1] Further observations were made on the distribution of the disease and data on its transmissibility were obtained since the publication of the first account. In view of the fact that no other record of the occurrence of the above-named organism has been found in the literature treating of sugar-cane fungi, and that the disease it causes is not distinctly differentiated by other authors from other similar troubles, it will be of interest to publish more fully the facts thus far known concerning this disease and to describe in greater detail its distinguishing features.

### ECONOMIC CONSIDERATIONS.

From observations made on sugar-cane diseases in Porto Rico it is now certain that in so far as reduction of yield is concerned this dry top rot or vascular disease is the most serious of the three or four major diseases of sugar cane existing at present in Porto Rico. It arrests and dwarfs the growth and causes the death of the growing tips of the canes, and in advanced stages, on account of the drying of the cane tops, the tissues already made and matured are speedily broken down by the fermentative action of saprophytic organisms which invade the stalk through the dead or wilted top. The disease was found distributed in practically all the principal sugar-cane growing section of the Island, on the north coast as well as on the irrigated south coast, in isolated areas on the western end, and in the central district around Cayey and Morovis. It was particularly noticeable in fields which showed evident signs of retarded growth and dwarfing especially in ratoon fields.

[1] Numbers in parenthesis refer to references listed at the end of this paper.

In times of low sugar prices the growers depend largely for their returns on ratoon crops which naturally can be made at a lesser cost than plant cane, but it is a well-known fact that a large proportion of our sugar-cane fields can not be ratooned more than two or three seasons, and these produce very poor crops even in their first or second ratoons. In examining minutely representative samples of dwarfed, abnormally thin, dry top rotted canes from different sections of the Island during the last three years, it became evident that *Plasmodiophora vascularum,* an organism which was only recently recognized as a cane parasite, is an important factor and in many instances the chief cause of not only ratoon failures but also of poor stands of plant cane, especially when infected seed were used or when healthy seed were planted in soils infected with the above-named organism. A reduction of 25 per cent from normal tonnage was estimated for a considerable proportion of fields on the north coast during the 1921–22 harvest on account of this disease. At Río Piedras a field of Cristalina plant cane which was infected with this disease produced an average of only fifteen tons of cane per acre. An especially poor stand of plant cane was seen near Barceloneta, where about 25 per cent of the stalks in a field of Rayada (purple stripe) cane were infected and dead by the time of harvest, and a similar field of plant cane was found on the south coast. In all of these cases numerous microscopic examinations were made of the affected canes and *Plasmodiophora vascularum* was constantly found inhabiting their fibers. It was evident that this organism was the principal cause of loss of canes in these fields.

This disease was found in our best varieties, Cristalina, Rayada, D–109, Yellow Caledonia, Otaheite and others, and it works in an obscure and at first unnoticeable manner until the canes are almost mature when the leaves become yellowish and begin to dry out from the interior of the top. Such phenomena may be caused by other agencies such as excessive infestation of insects, especially borers, lack of drainage, drought, and lack of plant food in the soil. At the present time all of these play their parts in reducing cane production in the Island, but this dry top rot as a disease is independent of either soil defficiencies or insect pests. It occurs even when the soil is in good condition and apart from insect injury. It may not be equally severe in different years, but the

last three years evidently were favorable for the propagation of the disease.

SYMPTOMS.

The pronounced visible signs of this disease in the leaves are, first, loss of green color, rolling in and withering or wilting, and lastly, drying of the tips of the central leaf whorls. (Fig. 1.) This is followed by the death of the points or the uppermost joints of the canes. The result of the disease is seen in the wilted and stunted condition of the affected stalks. The last-named phase of the disease occurs naturally only when the infection is severe. The dead top does not, as a rule, possess characters of a soft decay, and no particular characteristic odor has been noted from the decayed tissue. It is in reality a premature drying out of the tender and growing uppermost leaves and joints due to the clogging of the vascular system with a foreign organism. Upon removing the leaf sheaths of an affected cane stalk it is noticed that the internodes are gradually shorter and thinner towards the top and the entire shortened stalk possesses a marked tapering appearance resembling a tallow candle. (Fig. 2.) Healthy canes normally possess shorter internodes near their bases

FIG. 1.—The three canes in the center of this photograph show rolled-in, withered and dry tips of the central leaf whorls.

while the internodes of the center and top are usually longer and of nearly equal thickness throughout. In canes more severely affected the entire stalk fails to develope to normal length. (Fig. 3.) Often the drying of the leaves begins with one or more dead, gray, longitudinal stripes, of about 1 centimeter in width, at about the middle of the blades of the innermost leaves. It is often noted that here the premature drying and death of the younger inner leaves often begins not from the outer edges but from points in the middle of the blades along and near certain fibro-vascular bundles.

There is another more distinct and important internal symptom which is characteristic of this disease, and that is, the color of the fibers which appear to the unaided eye orange, yellow, sometimes pink, and even red. This coloration is due to the presence of the organism *Plasmodiophora vascularum* in the pitted vessels and annular tracheids of these fibro-vascular bundles, and it is usually confined to the lower and subterranean portions of the diseased sugar-cane stalk and roots. A microscopic examination of a thin section cut across one or more of these fibers shows the spherical orange-colored spores and the slightly yellowish, almost hyaline yet granular masses of the younger stages of *Plasmodiophora vascularum* clogging the water-conducting elements, vessels and tracheids, in the above-named fibro-vascular bundles. (Fig. 4, 6.) This symptom has not been recorded to be associated with any of the

FIG. 2.—On the left is a top of a healthy Cristalina cane stalk; on the right is a top of a diseased cane of the same variety and of the same age.

known sugar-cane diseases and it constitutes the most important feature in distinguishing and diagnosing this disease. Infected fibro-vascular bundles were found in several instances in the uppermost joints and leaf sheaths of cane.

The dry stripes in the inner leaves and the dry-top symptoms may not show until the disease is either well advanced or only when a larger number of fibro-vascular bundles are infected and clogged with the organism. The water content of the soils in which infected canes grow may have a direct influence upon the drying of the top leaves. During wet weather the external symptoms of the disease are not commonly noticeable, but as soon as the soil becomes dry the leaves turn pale green and even yellow and the characteristic drying from the tip or in stripes sets in. However, some cane stools which were grown from known infected seed were found to contain at the age of eight months, stalks, in addition to others showing top decay, which failed to show any abnormalities in their leaves, though these stalks were somewhat stunted and contained *Plasmodiophora vascularum* in some of their fibers. As a rule, the canes which show all the symptoms described above may be found to constitute only a small proportion of all the affected canes in a stool, but the few pronouncedly affected canes serve to indicate the presence of the disease, perhaps in a

FIG. 3. — Mature sugar-cane stalk underdeveloped, as seen from its small number of joints, due to Plasmodiphora vascularum.

not advanced stage, among the other canes in the same stool.

Stunting of sugar cane alone is not always a true indication of dry-top disease. There are other causes which may prevent growth in cane, but stunting, dry-top decay, and orange-pink coloration of otherwise uninjured fibro-vascular bundles of sugar cane constitute together the visible features of this disease which is distinct from any other sugar-cane trouble described from either Java, Hawaii, Louisiana, the Philippines, the West Indies and South America.

### SIMILAR PHENOMENA IN OTHER CANE DISEASES.

There are other fungi which if gaining entrance into the sugar cane give a red, vinous, purple or even black coloration to the affected parts. *Thielaviopsis paradoxa*, *Melanconium sacchari* and *Colletotrichum falcatum* in their invasion of cane tissue penetrate all elements of the stalk and produce a brown, vinous, red and sometimes black coloration in the fibers and the pulpy tissue. The fact that these fungi penetrate parenchyma tissue as well as fibers and that their mycelial threads are easily distinguishable in the cells differentiates these from the dry-top organism. In the case of the latter no decay is directly produced; the organism does not possess any mycelium and has not been so far observed in parenchyma or pulpy tissue surrounding the infected fibers. It inhabits the fibro-vascular bundles exclusively; the parenchyma tissue surrounding these remain unaffected and unaltered in appearance. Unlike the other

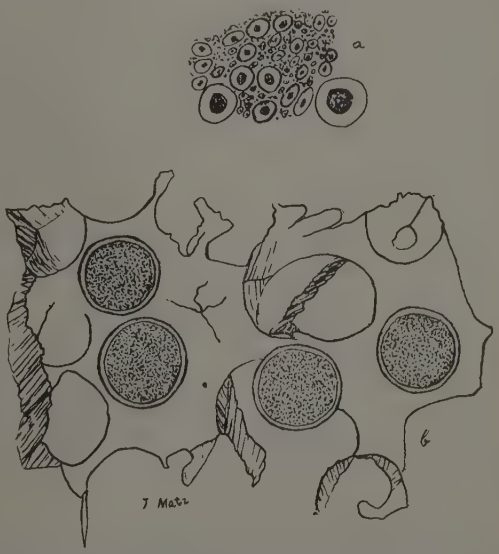


FIG. 4.—(*a*) Young stages of the spores of Plasmodiphora vascularum. (*b*) Mature spherical spores of the organism embedded in a broken gelatinous layer.

fungi it can not be considered a directly destructive agent. The damage which it causes to the cane plant in which it lives is only the result of interference through clogging of the vascular channels rather than actual breaking down of cells as is the case in most other obligate or facultative parasites. Thus it was found that canes in which the visible infection of fibers had not reached farther than the lowest three or four joints, yet the tops had already become dry and wilted due to the sole fact that the innermost fibers which feed the central leaves became clogged with the colonies of the organism at the base of the stalk. The partial death of leaves of sugar cane in the form of stripes parallel to the midrib does also occur in canes affected with gumming disease or moth stalk borer (*Diatræa saccharalis*). In these last two instances the dead stripes in the leaves may be traced to fibro-vascular bundles infected with *Bacterium vascularum* Smith in the case of gumming, and to severed vascular connections in cases of moth-borer injury. Therefore, in diagnosing the nature of dry-top decay of sugar cane in the field it is essential to examine for the borings of the moth borer and also for the more positive symptoms of gumming, which is the exudation of a yellow sticky mass of bacteria from the cut ends of the cane, in order not to confuse these with the symptoms of true dry-top rot. The last, however, occurs independently of any mechanical injury, or gumming disease. It may sometimes be mistaken for any one of the latter troubles when making casual examination of standing cane in the field, but a microscopic examination is necesary to determine whether the vascular *Plasmodiophora* is present in the infected fibers.

## HISTORY.

This disease has no doubt existed in variable quantities in cane fields here and elsewhere for some time in the past. It can hardly be considered a newly introduced disease into Porto Rico, because it is present practically in every sugar-cane growing section of the Island and in such varieties as Rayada, Crystalina and Otaheite, some of the oldest kinds grown here. It is true that it was found in Yellow Caledonia and D–109, canes of more recent introduction, but it has also been found in the roots and stalk of a seedling cane which was originated at Río Piedras, within the last four or five years. It is evident from field observations, and especially from the fact that it was found in the roots and fibers of the seedling cane mentioned, that the disease causing organism *Plasmodiophora*

*vascularum,* can be transfered from cane to cane through the soil, which may become infected with the above organism if diseased ratoons are allowed to decay in the ground. There is, therefore, the possibility that the organism might have been brought here in some new cane variety and that our older varieties became infected, by contact later, because of their strong susceptibility to this particular disease. Previous workers interested in sugar-cane diseases in Porto Rico have not recorded the occurrence of this vascular disease in sugar cane, though Johnston and Stevenson (2) mention a withertip of sugar cane which has appeared in Porto Rico during the years of 1911 to 1914 and name several fungi, *Hormiactella sacchari, Periconia sacchari* and *Colletotrichum falcatum,* found on the leaves of diseased plants. It is not stated whether search for organisms or other phenomena was made in the vascular bundles of the affected canes. The occurrence of the orange-pink and bright yellow-colored fibro-vascular bundles indicating the presence of *Plasmodiophora vascularum,* might have been overlooked due to the fact that no alteration in appearance in the surrounding parenchyma is caused, nor is the organism commonly found all through the stalk, the lowest joints only being infected in most cases, especially when the canes proceed from partially infected seed, which is perhaps the most common origin of the existing infections. The custom here is to use top seed, this has the advantage of eliminating the heavier infected portions of the cane stalk from being used as seed, and in this manner the distribution of the disease is kept down to its present proportions. Some heavily infected fields at least are known to owe their infections to the use of seed from old "run-down" ratoons; in other words, from canes in which the organism has most likely penetrated to the uppermost joints. It is of little importance to attempt to prove whether this disease was known to exist here in the past; such knowledge would be of little profit since the nature of the disease surely was not known. But what is of real value is to know its present distribution in the world, for should it exist at present in other sugar-cane growing countries there may also exist such controlling factors which might be applied here. Dr. E. J. Butler, of the Imperial Bureau of Mycology, in a letter to the writer stated that he did not see *Plasmodiophora vascularum* in India. Dr. L. O. Kunkel, of the Experiment Station of the Hawaii Sugar Planters Association, after having examined a mounted slide of *Plasmodiophora vascularum* in sugar cane tissue which was sent him, stated in a letter that

this disease does not occur in Hawaii nor has he seen the organism previously in his studies of *Plasmodiophoras.*

Not until the discovery of *Plasmodiophora vascularum* in the fibro-vascular bundles of the lowest joints in canes which were affected with Mosaic, and soon afterwards in non-Mosaic canes, was dry-top decay considered distinct from root disease. The rolling-up, withering and death of the top leaves of sugar cane was considered a symptom of root disease. This is true where root rot occurres, but dry-top rot appears on soils where root rot may not at all be present. Color in the fibers, such as the orange reddish of the dry-top rot, is not associated with what is usually considered as root disease. From the mode *Plasmodiophora vascularum* lives and grows in the interior of sugar cane, it is of a distinct nature from organisms which might cause root decay. There seems to be no true parasitic relationship in the form of host-cell destruction between this organism and the sugar-cane plant, since the vessels of the fibers though becoming filled with the organism do not suffer direct change, and in no case was it noted in the surrounding parenchyna cells. Apparently the organism is not able to break through cell walls. It may enter, however, through a broken or decayed root, and this is very likely the usual way the organism finds entrance into hitherto healthy canes. Canes which showed more than half of their number of fibers infected with the organism did not reveal any signs of decay in the parenchyma tissue. However, the phloem in some of the infected fibers was reddish and apparently dead.

### TRANSMISSIBILITY OF THE DISEASE.

Infected seed when planted may produce one or more infected stalks. These become infected directly through the fibers connecting the original buds with the mother seed piece, the organism passing directly to the new shoots through the continuous fibro-vascular system. As the fibers in the basal parts of the canes become filled with the organism the tops of the shoots begin to die as a result of blocking the food and water channels. Ordinarily the effects of the disease are not noticeable in young cane, since the growth of the organism is exceedingly slow, but in heavily infected seed a considerable number of young shoots die early.

Several trials to transmit the disease were made. The organism *Plasmodiophora vascularum* was teased out from the fibers and introduced into the root eyes of healthy seed. The resulting stools from these inoculated seed were stunted and presented symptoms

of dry-top rot but the organism was not recovered in the fibers of the stalks in these new canes.

Another experiment to prove whether infected seed are vehicles of transmission for this disease came out successful. Fifty Rayada seed were obtained from an infected plantation. These seed were examined with the aid of a microscope and razor-cut sections of these were found to contain numerous fibers infected with *Plasmodiophora vascularum.*

**TABLE SHOWING THE NUMBER OF STALKS AND THE PROPORTION OF INFECTED STALKS PRODUCED FROM SINGLE SEED INFECTED WITH PLASMODIOPHORA VASCULARUM.**

| Stools numbered in consecutive order. | Canes in stool. | Canes infected. | Stools numbered in consecutive order. | Canes in stool. | Canes infected. |
|---|---|---|---|---|---|
| 1 | 2 | 2 | 22 | 1 | 1 |
| 2 | 5 | 5 | 23 | 1 | 0 |
| 3 | 2 | 0 | 24 | 1 | 0 |
| 4 | 4 | 4 | 25 | 3 | 0 |
| 5 | 4 | 1 | 26 | 2 | 0 |
| 6 | 2 | 0 | 27 | 1 | 0 |
| 7 | 1 | 0 | 28 | 3 | 0 |
| 8 | 5 | 1 | 29 | 2 | 0 |
| 9 | 6 | 0 | 30 | 3 | 3 |
| 10 | 4 | 2 | 31 | 1 | 0 |
| 11 | 6 | 0 | 32 | 2 | 0 |
| 12 | 3 | 3 | 33 | 1 | 0 |
| 13 | 3 | 1 | 34 | 2 | 0 |
| 14 | 2 | 1 | 35 | 3 | 0 |
| 15 | 5 | 0 | 36 | 7 | 0 |
| 16 | 4 | 3 | 37 | 2 | 0 |
| 17 | 3 | 1 | 38 | 1 | 1 |
| 18 | 2 | 1 | 39 | 2 | 1 |
| 19 | 1 | *1 | 40 | 4 | 2 |
| 20 | 2 | 2 | 41 | 4 | 3 |
| 21 | 3 | 1 | | 115 | 40 |

* Infection in this stalk reached to a height of three feet from its base.

All the infected seed were planted singly in holes in land which was known not to have been planted to cane for many years. As a check 100 healthy seed of the same vaviety were planted in an adjacent plot alongside the infected seed. At the end of nine months all the canes were harvested and none of the healthy seed produced a single stalk which contained *Plasmodiophora* in its fibers while the diseased seed produced in 41 surviving stools only 115 stalks and 35 per cent of these were infected with *Plasmodiophora.* The stools from the healthy seed were normally developed in every

respect the number of canes in each stool ranged from four to ten and the canes attained a normal height. In the diseased stools the number and size of the canes were so reduced that it represented a very inferior crop for plant cane. At first when these seeds germinated there was nothing abnormal to be noticed. Numerous shoots were produced in the two lots of seed, but in the third and fourth month many of the younger shoots in the diseased seed stools began to dry up and die. The dead shoots did not yet reveal the presence of *Plasmodiophora* in their fibers at that time. Apparently the organism had not yet reached, in its development the interior of the stalks of these young shoots. In the seventh month, however, typical dry-top rot began to appear and *Plasmodiophora vascularum,* the same organism previously seen in the parent seed, was now noted in the fibers of their offspring stalks. From this experiment it became evident that diseased seed carrying the vascular *Plasmodiophora* transmit the disease to the next generation of canes. This mode of transmission is very likely the most usual one. The prevalence of the disease is proportional to the amount of its intensity in the fields from which the seed came.

Experiments bearing on the possibility of soil infection and the transmisibility from infected soils to healthy seed planted there are not terminated at the present time, but field observations were made with that point in view. At the Insular Experiment Station Cristalina cane, which were imported from Santo Domingo and which were noted for their vigor, were planted and produced three crops. During their second ratoon dry-top rot was noted in five stools at one corner of the field. That was the end of ratooning in that field, and it is being rested during this year. On the south coast a field of cane was examined and found to be heavily infested with dry-top rot. Numerous canes contained *Plasmodiophora vascularum* in their fibers. It was learned that cane planting failed there persistently in spite of renewal with vigorous seed from other parts. At present there is an experiment in progress on that field under the immediate care of Mr. F. S. Earle of Central Aguirre. The most conclusive case of soil transmission was noticed in 1919 at the Insular Experiment Station when *Plasmodiophora vascularum* was found in the roots and stem of a cane seedling which was originated only two or three years previously. It should be borne in mind that certain root-infesting insects might act as conveyors of the disease, but the disease has been found in uninjured cane.

There are therefore two possible means by which the disease can be transmitted and propagated. The surest way of its propagation is by the use of infected seed as was proved experimentally, and the probable way is through infected soils. The organism may become liberated from the decayed roots of infected stools and remain in the soil to be taken up again by the roots of the cane of the following planting. However, practically in all the severely infected fields, with the exception of the one field on the south coast mentioned above, which were examined, the infection was directly traceable to infected seed. As this is a vascular disease and the fibers being always enclosed by unbroken layers of tissue it is only reasonable to assume that the most likely entrance for the organism from the outside is through the soil and through roots which are likely to become decayed and broken.

## COMPARISON WITH OTHER DISEASES.

The "Sereh" disease of sugar cane, though quite distinct from anything we have in Porto Rico, has points of similarity to our dry-top rot, *i. e.*, in the occurrence of the red fibro-vascular bundles, in the stunting of canes, also that it is transmissible by diseased seed. From a recent colored illustration of Sereh disease in an article by Dr. Lyon (3) it is clearly seen that the red color in the fibers is localized mostly around the nodes and are not, as a rule, continuous through the internodes, while in the dry-top rot in Porto Rico the colored fibers, besides being only occasionally red or pink but more often orange-brown and yellow, are found mostly in the basal part of the cane and are continuous there through the internodes and nodes. In other words the colored vascular bundles, which are in reality not colored in themselves but contain an organism which possesses inherent color, are noticed in a region where the organism is most abundant, and that is nearer the root region or the mother seed which carried the original sources of colonies of the organism. The substance in the red fibers in Sereh is described as gum, while in dry-top disease it is distinctly granular and more often composed of larger spherical spores, with distinct walls and contents, embedded in hyaline or yellowish gelatinous matrix. This last substance is noticed with a microscope when an infected fiber is crushed and the compact mass of spores in the vessels is pressed out and broken up (Fig. 4.) Since Dr. Lyon has set forth so clearly the characteristics of Sereh it is evident that there is no disease in Porto Rico which can be diagnosed

as Sereh. However, in dry-top rot we find an analogous infectious dwarfing accompanied by a dry-top rot and associated with yellow to reddish colored fibro-vascular bundles.

Fiji disease with its characteristic galls is not present in Porto Rico. No swelling of tissue accompanies dry top rot.

Earle (4) in a discussion on sugar-cane root disease concludes that "Root disease as here understood is a complex including phases often known as 'Root Rot,' 'Wither Tip,' 'Top Rot' and 'Rind Disease.'" According to Mr. Earle, root disease is a collective name embracing phenomena which might be due primarily to an affection of roots by one or more obligate parasites or to defective cultural methods which enable the facultative parasites to attack the already weakened plants. The same author distinguishes the top rot in young overshadowed suckers, and the top rot caused by the moth borer (*Diatræa*) known as "dead heart," from that top rot which is due to root infection by parasites. Of these parasites there are those which attack the roots proper, such as *Rhizoctonia* and *Pythium,* and *Plasmodiophora vascularum,* which does not cause actual decay but clogs the water passages in the fibro-vascular system.

Gumming disease of sugar cane caused by *Bacterium vascularum* causes decay of the terminal part of the stalk. This is rather common now in Porto Rico, in fields where the Otaheite, Rayada and Cristalina canes are planted together. However, this disease is easily distinguished by the peculiar reddish spotting on the almost white and light-green areas in the top leaves in the early stages of infection and by the yellow gummy exudation of the cut surfaces of infected canes, symptoms which are not associated with dry-top rot.

Kruger (5) in his book mentions on page 336 an uninfections top rot which occurs here and there amongst healthy stalks in young cane. At first the young and yet unrolled heart leaves are attacked. These turn brown and die, then the older leaves begin to die as well. The vegetative point is attacked. It also turns brown and produces when cut an unpleasant odor. The disease works from the top down into the stalks, which finally dies. No specific cause is assigned. Kruger distinguishes this disease from the Australian and South American gumming disease and from the borer "dead heart," and suggests that it may agree with a disease of the same name described from Mauritius.

V. Gorkum (6) describes a top rot from Brazil which is accompanied by a crinkling and folding of the growing point as if

the innermost leaves were confined by an outer tightly enclosing envelope. This was noted here in a few rare instances but the plants soon recovered their normal appearance.

The drying of the top and resulting loss of tonnage of sugar cane in Porto Rico, which is the subject of this paper, refers only to cases where the initial withering or drying takes place without the direct action of microorganisms, insects or other direct agencies, on the tissues of the younger, inner leaves of the cane tops. In such instances where specific parasites cause directly the destruction of tissue they are recognized, whether they be insects, fungi, or bacteria, by closer observation. *Plasmodiophora vascularum* does not directly attack nor destroy the tissues of the cane stalk. It merely clogs the lower portions of the fibers, thus starving the top of the plant. Perhaps the nearest approach in point of similarity in behavior is the disease of sugar cane described by Butler (7) and by Dash (8) under the name of "wilt." The presence of the fungus *Cephalosporium sacchari* But., however, distinguishes that disease from the dry-top rot of Porto Rico. It is possible that the former is present here as well, but in diagnosing dry-top rotted cane no mycelium was found in the tissues of the stalk, neither was noted in them the hollowness of the stalk which accompanies wilt.

Cook and Horne (9) in their discussion of insects and diseases of sugar cane in Cuba mention a drying out of cane with the following symptoms: "Many of the stalks of these diseased plants have made an apparently good growth of several feet, but the leaves are thickly clustered at the top, the internodes having failed to elongate, and a rot is found passing down along the inner or younger leaf sheaths to the top of the stem." They further state "The more easily recognized common enemies of the cane are present, but their individual or combined effect is not sufficient to account for the drying out." Their illustration suggests a strong similarity to dry-top rot of cane as it occurs here.

In summarizing the above review of the recorded occurrence of top decay in sugar cane it is apparent that there exists a brown top rot or dead heart, causing discoloration and decay of the tender tissues of the tops of younger cane, as described by Kruger from Java, mentioned by Earle from Porto Rico and discussed by von Gorkum from Brazil. This decay is attributed to borers or overshadowing of young shoots and to other not fully determined physiological interferences. There are also bacterial top rots; one at least is caused by the gumming disease organism, *Bacterium vas-*

*cularum*, and it is probable that there exist other bacterial organisms capable of causing top decay in sugar cane; then there is wilt as described by Butler and Dash and which disease they attribute to the fungus *Cephalosporium sacchari* which attacks the interior of cane stems; and lastly we have wither tip as recorded by Johnston and Stevenson, and the drying out of cane described by Cook and Horne from Cuba. The last two diseases are perhaps the same as the dry-top rot, which is at the present time distributed widely in Porto Rico and which is the subject of this paper.

## THE DISTRIBUTION OF THE DISEASE IN PORTO RICO.

Our knowledge of the present distribution of the disease in Porto Rico is based on the finding of *Plasmodiophora vascularum* in the interior of the fibers of affected cane. The organism was first noted near the town of Bayamón, in the variety Cavangerie or Caña Colorada, a claret-colored cane with a dark-green stripe in the stem and sometimes with a yellow or white longitudinal stripe in the leaves. The cane did not show typical top rot, at least it was not noticeable. Later it was found in a Porto Rico seedling at Río Piedras; in Rayada at Río Piedras, Guánica, Cayey, and Salinas; in D–109 at Loíza, Toa Baja, and Morovis; in Cristalina at Mayagüez, Manatí, Barceloneta, and Arecibo; in Yellow Caledonia at San Germán and Cambalache; in Otaheite at Guayanilla and Mayagüez. In a number of occurrences it was most commonly found in Rayada. This, however, does not imply that this variety is the most susceptible. This cane variety is the most commonly grown in a large proportion of the cane fields of the Island, and because of the superiority of this variety in sweetness little discrimination is exercized in using it for seed purposes. Old diseased ratoons which are perhaps of no value for grinding are cut and used for seed, and thus the disease is propagated in this variety.

## THE SYSTEMATIC POSITION OF *Plasmodiophora vascularum*.

In my description of the organism in an article published in 1920 (1) the characters of the organism were described from material as it was found to exist in the cane plant. The outstanding features of the organism and its behavior may be summarized as follows:

1. The organism possesses a young stage in the form of a plasma or plasmodium composed of a hyaline or slightly yellowish granu-

lar mass of cytoplasm. This is noted to occur at the advancing point of the organism; that is, at the forward extreme of its penetrations in the fibers of the sugar cane. Development in this stage takes place through the enlargement of the individual granules. A gradual increase in size of these granules is noted towards its maturer or lower portions in the fibers of the cane. These maturer granules are composed of a densely granular center surrounded by a definite clear layer of cytoplasm of variable size depending on the age of the individual granule. This phase in the life of the organism resembles the plasmodia stage of *Plasmodiophora brassicæ* in the cells of its host, the cabbage. However, the granules of the sugar-cane organism cannot be considered as merely nuclei of the plasmodium because actual growth takes place in them. They develop from the size of a minute granule to a mature spore which may measure sixteen microns in diameter, therefore these bodies must be considered as individuals living together the same as the amoeboid bodies do in *P. brassicae.*

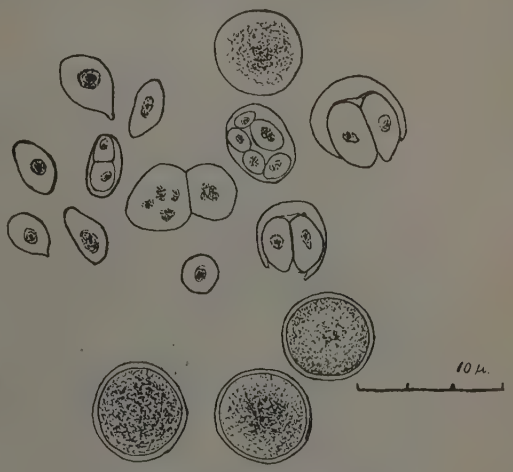


**FIG. 5.—Stages in the development of Plasmodiophora vascularum.**

In studing the behavior of *P. vascularum* it was observed that the individual bodies, which are composed of a granular center surrounded by a larger hyaline layer, undergo a process of division which is rather unique in itself. At first the central granule divides into two or four and sometimes six parts. Each subdivided granular portion becomes surrounded by a hyaline layer which grow until they force the outer layer of the mother granule to break and they are liberated. (Fig. 5.) This phase of growth finds no parallel in the described life phases of *P. Brassicæ.* It is hardly conceivable that these bodies should be nuclei and that this is their mode of division, because it was definitely observed that

these bodies gradually mature directly into large resting spores. They must therefore be considered as immature spores.

2. The mature stage or the resting stage of the organism is the

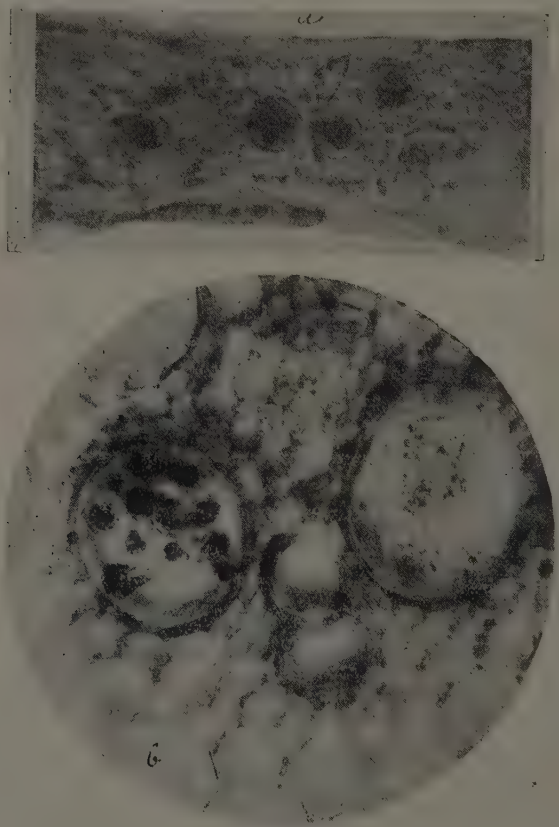

**FIG. 6.—*a*, Continuous mass of the plasmodium pressed out from a vessel of a sugar-cane fiber. *b*, A fibro-vascular bundle with the two stages of the organism in its large vessels.**

more commonly noticed in infected cane. It consists of comparatively large, spherical smooth spores which are granular or sometimes coarsely granular in the interior with somewhat thick but

hyaline walls, and are yellow, orange, sometimes slightly brown in color, measuring around .014–.016 millimeters in diameter. These spores are imbedded in a clear or somewhat yellowish, at length hardened homogenous matrix. At this stage the organism resembles the well-known Physoderma zeae-maydis in the cells of the corn plant. However, the sporangia of the latter are considerably larger and are not spherical but flattened on one side. The mode of germination of *Physoderma* as illustrated by Tisdale (10) has not been observed in the sugar-cane organism. No mycelial connection of any sort was observed in connection with the latter. Another point of distinction between the two is that *Physoderma* lives in the parenchyma cells of its host, while the sugar-cane organism has so far been found exclusively in the vessels of the fibers. In this respect it also differs from *Plasmodiophora brassicæ* since the latter occurs in the parenchymatous tissues of its host. However, the sugar-cane organism has after many attempts refused to grow outside of its host, the sugar-cane stalk, and no success was obtained in the many efforts to germinate its resting spores for that reason the exact relationship of this organism cannot be definitely placed. The fact is that one of its stages is a free *Plasmodium* limited by the walls of the vessels of the host and it thus possesses a principal character of the *Plasmodiophoraceæ*.

Dr. Elliott described a new organism, *Cystospora batata*, as the cause of sweet-potatoe soil rot or pox (11). In comparing some of his illustrations of that organism with the different phases of the life history of the sugar-cane organism some very strikingly analogous figures are noted. The cysts of *Cystospora batata* are admirably resembling the resting spores of *Plasmodiophora vascularum*, but there is a radical difference in the mode of formation of the two. According to Dr. Elliott the cyst does not gradually grow into its final shape. He states the "plasmodium which fills the cell becomes dark and heavily granular. It gradually contracts into a dark dense central mass surrounded by a clearer zone which becomes a thick clear wall surrounding the central neclear material." Evidently the cyst does not undergo growth as does the resting spore of *P. vascularum*. In his illustration of stages in the growth of a plasmodium Dr. Elliott figures an individual plasmodium with sixteen nuclei which resembles the spore-forming stage of the sugar-cane organism, but instead of designating the smaller bodies within the plasma nuclei these are in the sugar-cane organism really spores in the process of formation, because all the stages

from the minutest granule to the completely developed spore are to be seen in the same continues vessel in the fibers of the cane. Dr. Elliott concludes that his organism undoubtedly belongs to the *Plasmodiophorales,* but because of the formation of the heavy-walled cyst with its large number of spores separates this plasmodium from *Plasmodiophora.* The behavior of *P. vascularum* is indeed not like *P. brassicæ,* nor is it like any of the *Plasmodiophorales,* especially when its partiality to the interior of cane fibers is considered. But it possesses no marked feature which would throw it definitely into another group.

## SUMMARY.

Dry-top rot of sugar cane is a distinct disease in Porto Rico, and is constantly associated with *Plasmodiophora vascularum.*

The disease occurs generally all over Porto Rico, but its existence outside of the Island has not been recognized.

The damage which this disease causes is heavy when diseased seed are used. Infected soil may also contribute to the failure of the crop, since it is possible that the disease may be transmitted to healthy seed planted in lands where the disease existed previously.

The disease has been transmitted experimentally by using seed which were naturally infected with *Plasmodiophora vascularum.*

To control this disease it is of primary importance that seed selection should be practiced. No seed should be taken from fields which have diseased cane; at least only topmost seed should be selected.

The entire length of the stalk is not always infected. Usually only the lowest or basal portions of the canes are infected, but if the disease has the opportunity to extend itself as in old ratoons of infected plantings where the canes are comparatively short, even top portions of these canes contain the disease-causing organism in an active stage and no seed should be taken for planting from such fields.

As a measure of precaution it is advisable to use seed from six or seven months' old stools, in which the disease did not have the opportunity to work up, provided the stools themselves come from healthy seed.

Soils, which are known to have produced heavily infected crops should be rotated to legumes or other crops, in order to starve out the disease-causing organism, since it appears that the sugar plant is, so far, its only host.

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